accomplished in any case that has not passed over the boundary between neurasthenia so-called and genuine insanity, and even in the early stages of the latter, as Case I would appear to show. The application of the truth requires personality and patience.

Reasoning backward, it would seem more and more probable, if normal conditions and health can be regained by the employment of active hygiene, embracing every measure possibly included in the term, and by hygiene administered against the patient's desire and by muscular force when necessary; if, I say, this be true, and I believe it to be a fundamental truth, then it would seem fair to conclude that neurasthenia is a misnomer and "hygiene hunger" the proper term. In one nerve exhaustion, in another trauma, in the next sexual excess, in a fourth that auto-intoxication peculiar to the disposition which Shakespeare characterizes when he speaks of those whose "fat blood slips lazily along their veins," in a sixth or seventh malaria or chlorosis, or overexertion of mind or body during rapid growth and development—any of these may underlie and cause, if you please, the symptom-complex that we have been content to denominate neurasthenia. Rest one, exercise another to the limit, feed those who need food, and starve the overgreedy; in every case provide a change of daily *regime*, overdose with clean water and clean air inside and out; and the time will arrive for everyone in which graduated exercise in the open air to the point of physical tire will begin to be possible and will serve as the road to health of mind and body.

Perhaps in the results that shall be obtained the physician will enjoy a renewed confidence in the justification of his existence, while the surgeon will sadly witness the reclaim of a wide range of purely medical conditions which he had dreamed might become his eminent domain.

NERVOUS MANIFESTATIONS OF ARTERIOSCLEROSIS.

By Alfred Stengel, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

Various nervous symptoms may be met with in cases of arteriosclerosis, some of them being certainly the result of the vascular disease, while others are due to associated organic diseases. It must be recalled that persons suffering from arteriosclerosis usually have some form of structural disease affecting the kidneys, heart, brain, or other organs, or several of these at once, and it is important to distinguish the symptoms caused by such associated conditions from those which are properly attributable to the arteriosclerosis as such. In a certain sense the nervous manifestations resulting from secondary organic diseases are, of course, assignable to the vascular disease that caused the organic trouble; but it is necessary to differentiate as far as may be possible between symptoms that result directly from the arteriosclerosis and those which have some intermediate organic derangement for their cause. More especially is it necessary to avoid the error, perhaps a little too likely to become prevalent, of attributing to arteriosclerosis, various symptoms that arise as a result of purely accidental organic diseases associated with the vascular trouble but not essentially due to it. The same difficulty occurs here as has happened in the past in the cases of such general conditions as neurasthenia, hysteria, anemia, etc. It is easy to acquire the habit of regarding all sorts of symptoms as the result of such conditions and of overlooking associated diseases. The danger is even greater in the case of arteriosclerosis, which is so nearly universal among the aged that the indolent diagnostician may readily fall into the way of attributing all of the generalized and more or less obscure conditions of senility and even of the presenile period to arteriosclerosis. For these reasons I shall take pains in the following discussion to point out, first, the certain results of vascular occlusion or disease, and, second, the symptoms associated with cases of arteriosclerosis that may be fairly attributable to that condition by reason of their analogy with the symptoms included in the first group and by the exclusion of other probable causes. Increase of knowledge may hereafter establish more accurate methods of determining the relationship of certain symptoms to disease of the bloodvessels and their occlusion, but for the present we must rely upon the plan suggested.

SYMPTOMS ASSOCIATED WITH OBSTRUCTION OF PERIPHERAL VESSELS. The sudden occlusion or obstruction of one of the larger peripheral arteries is attended with immediate and usually quite characteristic symptoms. This may be observed when a larger artery is compressed with the fingers or a tourniquet, or when obstruction is caused by a large embolus from the heart or aorta There is sudden pain, often excessively violent in character, and generally loss of power in the parts supplied by the obstructed vessel. The pain may subside in a short time, to be renewed with secondary embolisms farther toward the periphery, or with increase of the direct compression; but later the violence of the pain is lessened, and instead of intolerable suffering a more continuous aching follows or the patient complains of darting pains of relatively moderate intensity extending from the place of obstruction to the extremity of the affected member. The loss of power is rarely complete and becomes less marked after the initial violence of the attack has subsided. In some instances, however, the paralysis is absolute. Thus, in cases of total obstruction of the abdominal aorta by an embolus there may be sudden and complete paraplegia, besides intense pains in the legs of the character just described. In addition to the two predominating symptoms, pain and loss of power, there are a number of others of a less conspicuous kind. Among these are pallor, coldness (objective and sometimes subjective), tingling or numbness, weakness or complete cessation of the pulses in the peripheral vessels, occasionally violent cramps of muscles, and sometimes lividity or edema. The tendency to cramps or muscular spasm was illustrated by the violent intestinal spasms produced in Thoma's experimental compression of the abdominal aorta. The other symptoms, more or less pronounced in severity, have been repeatedly caused by experimental compression of peripheral vessels and by embolism. Eventually the result of complete occlusion of a large artery is recovery by establishment of a collateral circulation or gangrene.

The following cases illustrate the direct association of pain, loss of power, etc., with occlusion of the main artery of a limb:

J. W., aged seventy years, had advanced general arteriosclerosis and myocarditis. He had two attacks of embolism, or more likely cerebral hemorrhage, each, rather remarkably, affecting the cuneus region. After the first there was hemianopsia, after the second nearly complete blindness. Finally, in June, 1907, there occurred a sudden embolic occlusion of the right external iliac or femoral artery, attended with such intense pain that the patient, though semi-unconscious, cried out in agony, and could be only partially relieved by large doses of morphine. The right leg became motionless at once, and subsequently recovered very little power. The posterior tibial and dorsalis pedis pulses could not be felt; the feet and later the whole leg grew pallid and cold. Two days later a similar attack occurred in the left leg and caused paralysis of the left leg, almost completing a paraplegia. Fortunately the patient did not long survive the development of these intolerable conditions, for the pain, though less marked than in the beginning, did not wholly subside, and every attempt to move the legs caused intense suffering and spasmodic contraction of the muscles. There were numbness and anesthesia of the skin, and before death necrosis of the heels and toes had begun.

A. V., aged fifty years, had an embolic obstruction of the femoral artery while walking. There was sudden pain in the back of the thigh, and the patient cried out, thinking that someone had struck him across the thigh with a stout cane. He turned round to see who had done so, when the power in the leg failed and he fell to the ground. This case is of interest because of the character of the pain complained of. In several instances within my knowledge the patient has expressed the same feeling of having been struck.

PAINFUL SENSATIONS PRECEDING SENILE AND DIABETIC GANGRENE. In these conditions vascular obstruction is less immediate and less complete than in the group just described, but it is unnecessary to offer proof of the well-established fact that the gangrene of diabetics and senile arteriosclerotics is preceded by gradually increasing obstruction of the arteries. Neither is it necessary to describe in this place the various symptoms, such as coldness, numbness, tingling, darting pains, and finally the intolerable suffering that accompany the conditions. It is, however, important to consider whether these various symptoms are directly attributable to the vascular obstruction or rather to some associated condition. Kussmaul and later Joffroy and Achard have described cases of neuritis which they regarded as resulting from arteriosclerosis. While this possibility is less applicable to the suddenly developed pains attending the onset of senile and diabetic gangrene than to the more gradual conditions to be described in the next group, it may be mentioned here as a suggested explanation of certain painful conditions associated with arteriosclerotic occlusion of the circulation In view, however, of the sudden intense pain of embolic or experimental obstruction, and of the continuing darting pains following the more severe onset, it may be doubted whether the intervention of a neuritis is a frequent, not to mention a necessary, condition.

In the same group as the cases of gangrene may be placed cases of intense cardiac pain due to occlusions of the coronary arteries. It is a well-known fact that in many cases of angina pectoris embolic or thrombotic occlusions of the coronary arteries and degenerative lesions of the myocardium are encountered, and it has been believed by some that the attacks themselves may be the immediate result of such sudden occlusions. In proof of this view cases have been reported in which after a very severe attack of angina leading to sudden death recent embolism or rapidly developed thrombosis of the coronary vessels has been discovered. It is not my intention here to suggest that such vascular occlusion explains all cases of angina pectoris, but it seems fairly certain that some of the cases of intense cardiac pain leading to a fatal termination have this causation. It may be well to recall in the same connection the intense pain sometimes met with in the back in cases of renal embolism and the similar pains found in the region of the spleen upon the development of a sudden splenic infarction.

PAINS AND OTHER NERVOUS SYMPTOMS IN THE EXTREMITIES LESS CERTAINLY ATTRIBUTABLE TO ARTERIOSCLEROSIS. A variety of nervous symptoms, including numbness, tingling, a sense of coldness or of heat or burning, and other forms of paresthesia (acroparesthesia), and sometimes moderate or severe pain, together with a sense of stiffness, and perhaps actual stiffness or weakness, occur in persons of advancing years with more or less arteriosclerosis. These symptoms more frequently affect the legs than the arms, although the paresthesia is not infrequent in the hands. The severity of the manifestations may have a direct relation to the degree of arteriosclerosis, and may increase in direct proportion with the advance of that disease. In other cases it may be observed that the symptoms increase and decrease as the condition of the heart suffices or fails to maintain the circulation in the sclerotic vascular system. As patients presenting these symptoms usually have other disorders in addition to arteriosclerosis, it may sometimes be doubted whether the nervous manifestations referred to are the result of the disease of the bloodvessels or of other (associated) conditions. The fact that the further progress of such cases shows only an increase in arteriosclerosis and its undoubted consequences, and not more pronounced local or general manifestations of other diseases, is rather convincing that the symptoms may be properly attributed to the vascular disease. Two cases of this sort may be very briefly cited:

J. R., aged seventy-two years, a highly arteriosclerotic man with associated cardiac hypertrophy, has suffered for some time with what he calls neuritis in his legs. There is tingling, burning, and pain in both legs, but principally in the right, from the knees to the ends of the toes. The pains are indefinite in location and cannot be increased by pressure along any of the nerve trunks. There is general weakness of the legs, but no definite palsy. The feet are cold, and at times become livid in spots. When his circulation is improved by the use of digitalis, strophanthus, and glonoin the pains decrease and in general his suffering is directly proportioned to his condition and especially the state of his circulation.

Mrs. R. S., aged sixty years, is markedly arteriosclerotic and has some gouty change in the joints of the hands. For several years she has complained of constant numbness and painful tingling in the fingers. To a less extent the feet have been similarly affected. There have never been acute gouty attacks. She has had no definite indications of neuritis.

In both of these cases the question may be properly raised whether or not the symptoms are the result of neuritis. The possible occurrence of an arteriosclerotic form of neuritis has already been alluded to, while in the second of the two cited cases another possibility, that of gouty neuritis, presents itself. In the first case the intensity of the symptoms has been so directly proportioned to the adequacy or inadequacy of the circulation that there has seemed little doubt about the directness of the association. In the other case, and this is but one of a number of similar type, one should perhaps be less certain of the relationship.

Allusion may now be made to a large group of more or less painful affections of the extremities associated with arterial disease in which the symptoms suggest the intervention of reflex vasomotor conditions between the arteriosclerosis and the acute manifestations. Among these affections are erythromelalgia, Raynaud's disease, "dead fingers," and intermittent claudication. All of these conditions occur in persons who have more or less decided arterial disease. In the first three the nature of the attacks and the paroxysmal character of the vasomotor phenomena suggest that reflex nervous influences play an important part in the phenomena, but the association of marked arteriosclerosis and the constantly recurring circulatory disturbances suggest that the reflexes have their origin in the arterial disease. So far as intermittent claudication is concerned, this peculiar syndrome has been generally accepted as due to arteriosclerosis since Charcot's original communication and Erb's more extensive studies. The symptoms do not differ greatly from those first referred to in this group, but the pains, paresthesia, stiffness, cramps, and weakness are distinctly the result of effort. It has been assumed by some that these manifestations result directly from the inadequacy of the supply of blood, when the exercise of muscular effort taxes the circulation, while others have thought that vasomotor spasm results from the exertion and causes the symptoms.

It has been pointed out in proof of the first of these views that painful cardiac attacks associated with coronary disease lessen when the myocardium has grown considerably sclerotic and when in consequence the need of active circulation is less imperative than in the case of a more normal heart muscle. Erb and some more recent neurologists have maintained that a nervous element plays a part by exciting vasomotor spasm, and in proof of this cite the facts that coldness, pallor, and weakness of the pulses are frequent symptoms of the attacks, and that claudication is especially likely to affect arteriosclerotic persons who have a general neurasthenic condition as well. On the other hand, it is significant of the importance of the purely obstructive character of the vascular trouble that claudication has been universally recognized, since Charcot's description, as a forerunner of gangrene.

Dejerine, Sollier, and others have described cases of intermittent claudication in which the symptoms have been practically the same as those usually recognized, but with the distinctions that the signs of peripheral arteriosclerosis (coldness, weakness of pulse, etc.) have been wanting and the knee-jerks have been normal or absent during rest and excited after exertion. In these cases there was found arteriosclerosis of the spinal vessels, and Dejerine attributes the symptoms to anemia of the cord. The following case resembles Dejerine's variety in the behavior of the reflexes, but there was a very decided arteriosclerosis of the peripheral vessels:

L. H., aged fifty-three years, height six feet, first consulted me about eight years ago, when he had chronic pharyngitis and beginning deafness which was found to be due to middle ear disease. Even at that date he complained of a peculiar form of weakness in the legs after exertion. He was admitted to the University Hospital seven years later with more marked symptoms, which then took the form of rather distinct claudication. After every exertion he suffered with weakness, cramps, and pains in the legs, and his general condition was one of increasing debility. He had arteriosclerosis of a decided grade involving the arteries in a uniform degree, and there was also distinct sclerosis of the peripheral veins. His station was slightly ataxic and the gait was somewhat shuffling, but after exertion, as in walking, he became notably weak in his legs, so that he could scarcely stand erect, and he complained of a sense of stiffness in his muscles. There were constant vague, indefinite pains in the legs, but no alteration in thermal or tactile sensation. The knee-jerks were usually absent, even on reinforcement, but after exertion or when under slight excitement the patellar reflex was present and sometimes actually exaggerated. He remained under observation for several months, growing more feeble as time went on, and finally became bed-ridden. His heart, which was a little hypertrophied and evidently deficient in power, failed, and finally, after a fall, he developed gangrene of one arm and died. The symptoms were those of intermittent claudication excited by exertion, but there was the possibility of a central (spinal) lesion suggested by the usual absence of patellar reflexes and by a tendency to vague sensory disturbances affecting the body in the region of the hips. My colleague, Dr. Spiller, inclined to regard the symptoms as sufficient to suggest a spinal lesion, but my own observations led me to believe that the condition was mainly, if not wholly, peripheral. We both agreed that the arteriosclerosis, whether operative peripherally or partly centrally as well, was the fundamental cause of the peculiar nervous manifestations in the legs.

In some cases the muscular stiffness and pain occur under precisely opposite conditions. In these instances, after sitting or otherwise resting, the patient finds, on attempting to rise to his feet or to walk, that the muscles have become stiff and painful and exertion is attended with more or less suffering. While in minor grades a common symptom in old people, this association occasionally reaches such a marked degree that it constitutes a clinical condition of much importance.

The diagnosis of this peripheral condition is sometimes extremely difficult, particularly in instances in which pains radiating through the limb suggest the existence of a neuritis. A number of such that have come under my observation have been so regarded. Among the distinctions which have impressed me as important are the absence of tenderness along the course of the nerves, the absence of anesthesia, and the presence and more or less proportionate grade of arteriosclerosis associated with the conditions.

ABDOMINAL PAINS IN ASSOCIATION WITH ARTERIOSCLEROSIS. Sudden obstruction of the abdominal aorta by a large embolus from the heart is known to produce intense pain; aneurysms of the abdominal aorta also occasion marked and often excessive pain; but there are conditions in which less serious disease of the abdominal aorta or other abdominal arteries may occasion painful conditions in the abdomen. It is not, I think, sufficiently recognized that a tortuous sclerotic abdominal aorta or a simple pulsating abdominal aorta when slightly sclerosed may occasion intense paroxysms of pain. Some of the cases of continuous backache in old people have impressed me as belonging to this group. In these it is difficult to prove the cause of the pain, but in some others intense tenderness of the violently throbbing aorta in an emaciated person in whom the vessel can be palpated indicates the direct relationship between the vascular trouble and the clinical condition Furthermore, the association of pain with atheroma of the thoracic aorta furnishes an analogy that is of some significance.

Pains in Arteriosclerosis of the Thoracic Aorta. Not rarely in individuals with marked sclerosis of the arch of the aorta painful paroxysms occur upon slight exertion. In some instances these have the definite paroxysmal character of angina pectoris, even though the coronary vessels have not been involved Usually, however, the pain is more diffuse, extending almost equally to the right as well as to the left shoulder or arm, and the relation of the attack to muscular effort is a closer one than in angina from disease of the coronary vessels. It has been suggested that sensory end-organs (Pacinian corpuscles) in the wall of the aorta explain the tendency to these phenomena.

Cerebral Manifestations of Arteriosclerosis. The direct relation of arteriosclerosis of the cerebral vessels to cerebral hemorrhage and thrombosis needs no discussion, but it may be well to consider here the possibility that many rather indefinite cerebral manifestations in persons suffering from arteriosclerosis may be the result of (partial not completely obstructive) thrombosis of larger cerebral vessels or of complete thrombosis of finer branches. Lesions of this sort are apt to be overlooked even by the pathologist unless his attention is directed especially to this point. The studies of MacCallum have shown how hyaline thromboses of the smaller cerebral vessels may occasion marked cerebral symptoms in malaria, and similar lesions not infrequently occur in arteriosclerosis. Partial thromboses of the larger cerebral vessels are frequently met with, indeed are rarely completely wanting, in cases of extensive arteriosclerosis of the circle of Willis and of the other cerebral arteries. These thrombi, though not completely obstructive, cause a rather rapid encroachment on the lumen of the vessels, and thus interfere with the circulation in a manner more abrupt and violent than does the narrowing of the lumen that results from increase of the arterial disease itself. It is not unlikely, then, that the symptoms usually attributed to obstruction of the circulation by increasing arteriosclerosis are often the result of such thrombosis. However this may be, the more important fact to establish is that certain cerebral symptoms suggestive of other conditions may be due to arteriosclerosis with or without partial thrombosis.

The influence of obstruction of the cerebral circulation in the development of symptoms is shown by the unequivocal results of complete thrombosis of large cerebral arteries, but even more directly by experimental studies, to which reference may now be made.

EXPERIMENTAL AND PATHOLOGICAL EVIDENCE OF THE INFLUENCE OF OBSTRUCTION OF THE CEREBRAL CIRCULATION IN THE DEVELOPMENT OF CEREBRAL SYMPTOMS. From the days of Avicenna, who caused loss of motion and sensation in animals by ligation of the carotid arteries, to the present time a long line of physiological experimenters have described a group of cerebral symptoms that could be caused by compression or ligature of the carotid vessels. Miller caused, in each of eighteen experiments, disturbed breathing and heart action, depression of temperature, sleepiness, vertigo, blindness, strabismus, spasms, and convulsions. Kussmaul and Tenner, in a more elaborate investigation, confirmed these findings and add results, exactly like those obtained in animals, which they obtained in human beings. They quote the results attained by a number of previous investigators, all of whom caused marked symptoms falling short of actual convulsions, and then cite their own experiments, in which convulsions were readily produced in six rather weak persons.

Naunyn was the first to call attention to the clinical occurrence of convulsions in persons suffering from cerebral arteriosclerosis, and his pupil Lucas[1] collected from the literature seven very striking cases in which convulsions (preceded by less severe nervous manifestations like those mentioned above) occurred in persons of middle or advanced age as the result of occlusion of the carotid arteries by thrombosis or compression by aneurysms and tumors. A study of the history and pathological conditions in these cases leaves little room for doubt that the cerebral symptoms were the direct result of the obstructed circulation. Lucas then reports two cases under the care of Naunyn in which elderly individuals with marked arteriosclerosis suffered from attacks of dizziness, headache, twitching of muscles, and great weakness. In one of the cases compression of the carotid arteries caused the rapid development of an attack in which the symptoms just mentioned, as well as temporary unconsciousness, occurred. The conclusions reached by Naunyn that these epileptiform seizures resulted from the conditions of the cerebral vessels appears to me entirely warranted.

Several cases of this sort have come under my observation, of which the following merit mention:

Mrs. W., aged seventy-eight years, has been under my observation for several years past. She has markedly sclerosed vessels and some hypertrophy of the heart. The kidneys seem reasonably normal.

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[1] Inaug. Diss., Strassburg, 1894.

There are occasionally a trace of albumin and a few hyaline casts, but the conditions do not appear abnormal for one of her years. She has had a cataract in both eyes, one of which was operated upon with entire success three years ago. When she first came under my observation, three years ago, I was informed that for some little time past she had had attacks of peculiar character, often at night and sometimes also during the day. These had been regarded as petit mal. They consisted of momentary loss of consciousness or bewilderment, followed by slight clonic convulsions affecting the arms and legs and not beginning in any particular member, so far as could be discovered, and ending with a period of mental confusion and aphasia lasting from a few minutes to an hour, and ending in a deep sleep from which she awoke to her previous condition. Such attacks had occurred with some regularity for a year or two, when she had during the summer a seizure which appears to me to have been cerebral embolism or thrombosis, and left her with moderate hemiplegia. When I saw her in the following autumn the palsy had to a large extent disappeared. She has since had a number of attacks of the same character. Like the earlier ones, these involve no special part, being quite as likely to occur on the well side as on that which is partly paralyzed.

Mr. A. J. O., aged fifty-five years, had begun to suffer with circulatory disturbances, on account of which he first consulted me three or four years before his death I found an advancing arteriosclerosis, which steadily progressed up to the time of death. About two years before his death, when he was in rather good condition, he was seized with a sudden convulsion while playing at golf and was carried home. I found a slight right-sided hemiplegia, involving the face and arm, which cleared up almost completely within six or eight hours The upper arm alone seemed involved, the power in the fingers and the forearm being more or less completely preserved. After a few days he had no evidence of weakness in the right side. During the followingsummer he had two convulsions while in Europe, and there consulted Marie and other neurologists, all of whom agreed in regarding the convulsions as "functional." After his return to America several other convulsions occurred at intervals of a few months. There was no specific history, and up to the time of his first attack he had never had a lasting albuminuria, though he once or twice had a faint trace of albumin in his urine. After the onset of the paroxysms his general health grew worse and his circulation steadily grew weaker. Toward the end of his life he developed evidences of renal disease, which impressed me as being directly the result of the failing circulation. There was nothing in the paroxysms at any time suggestive of a uremic condition, and after the original one the recovery from the paroxysms was almost immediate, the whole seizure lasting little more than five to ten minutes.

J. T., aged sixty-five years, a patient in the Philadelphia Hospital, had extreme arteriosclerosis and an enlarged but weak heart. The peripheral circulation was very poor. Extremities were usually cold. The patient's general condition was one of marked enfeeblement. The kidneys apparently were not diseased; at most, some senile change could be recognized. Suddenly, without premonition, a convulsive seizure involving the muscles of the right side of the face and neck and of the deltoid and trapezius of the right side developed while I was examining the patient. It was difficult to determine whether there was complete loss of consciousness, on account of the patient's general state, but there was evidently at least a partial unconsciousness. After a duration of about five minutes the attack passed off and the patient regained his previous condition. Subsequently he improved somewhat under rest and care in the ward, and he was discharged from the Hospital.

These three instances represent definite convulsive attacks in arteriosclerotic patients without any other explanation of the seizures. In two of the cases there was very probably thrombosis in at least one of the attacks; but the other attacks seemed to occur without any of the usual evidences of complete occlusion by thrombosis or embolism. In the third case there was but one attack, and this was not discoverably due to thrombosis. In view of the experimental evidence before cited, it may be regarded as established that sudden interference with the circulation, whether due to thrombosis or embolism, or independent of these accidents, may account for seizures such as occurred in the three cases just described. In the cases in which thrombosis or embolism are absent weakness of heart action may be the cause of the suddenly developed inadequacy of the cerebral circulation, while in other cases vasomotor spasm may possibly occur. The last explanation may apply to the seizures attending paroxysms of Stokes-Adams disease.

Stupor and Coma Associated with Cerebral Arteriosclerosis. The cases included under this heading are a little less definitely ascribable to the influence of the arterial disease alone, for the reason that the symptoms are less distinctly paroxysmal, and therefore less certainly the result of a temporarily active cause, such as circulatory disturbance. Nevertheless, in view of the fact that both stupor and coma have been produced experimentally by compression of the carotid arteries, and that in some cases the stupor suddenly appears and as suddenly disappears, the connection of the attacks with circulatory disorder does not seem unlikely. If one studies the symptomatology of extreme senility, there will be found a constantly recurring picture of stuporous states alternating with periods of mental alertness often quite surprising. The aged patient without warning falls into a lethargic or semicomatose state, and may remain so for hours or even days, and not infrequently wakes from this condition suddenly and completely, only to relapse again and again. The cause of these attacks may doubtless be an incomplete uremia in some cases, or other forms of toxemia, but the sudden relief of symptoms that so often occurs has convinced me that the attacks are more usually dependent upon the condition of the cerebral circulation.

Sometimes the attacks occur in persons who have not yet reached the period of advanced senility, and the attacks may be more lasting than those just referred to. In such cases a diagnosis of uremia is very likely to be made. Not a few of the cases of "uremia" occurring in such hospitals as the Philadelphia Hospital, where aged derelicts abound, are I think not actually instances of uremia, but rather cases of coma due to disturbed cerebral circulation the result of arteriosclerosis. One instance of this sort will suffice for illustration:

J. C. A., a man aged sixty years, was admitted to my ward at the Pennsylvania Hospital in complete coma. There was a high grade of arteriosclerosis and extremely feeble heart action. There was no albuminuria at this time nor subsequently while in my care. On two previous occasions he had been treated at the Pennsylvania and another hospital, and had been regarded as uremic. I was impressed with the intense weakness of his circulation and the extreme arterial disease, and advised against any depletive measures or other treatment such as is employed in uremia. Hypodermics of strychnine followed by administration of strophanthus and nitroglycerin by the mouth cauased rapid clearing of the stupor in direct proportion with improvement in his peripheral circulation, and twenty-four hours after admission he sat up in bed, took his food, and was generally quite decidedly clearer. Subsequently he lapsed somewhat, and his mental condition never wholly cleared up. A month later he was transferred to the Philadelphia Hospital, where his condition became less satisfactory, being one of extreme prostration and partial dementia. He was not easily manageable in the medical wards, and was transferred to the Insane Department, where he subsequently died without further developments. The records of his two earlier attacks are very fragmentary, not even the urinary examination being preserved. The diagnosis of uremia appears to have been made because the patient was comatose. In the last attack, which never cleared entirely, the duration was sufficient to have furnished some definite evidence of renal disease and uremia, had either been present, but repeated examinations of the urine failed to show any such evidence.

VERTIGO, TEMPORARY APHASIA, OCULAR DISTURBANCE, AND TINNITUS WITH CEREBRAL ARTERIOSCLEROSIS. All of these symptoms will be recognized as occurring in the group of conditions caused by compression of the carotid arteries and also among the manifestations of gross cerebral thrombosis. When one or the other occurs as a more or less isolated symptom in a case of arteriosclerosis, one hesitates to regard it as due to this cause alone, and very properly inclines to think it a part of some more definite focal, cerebral, or visceral condition not yet fully developed. In many instances further symptoms may occur, and a cardiac, renal, gastric, aural, or local cerebral cause may be recognized. Sometimes, however, such symptoms recur repeatedly and no further developments take place until perhaps with increasing cerebral sclerosis more distinct results follow.

PSYCHIC MANIFESTATIONS OF CEREBRAL ARTERIOSCLEROSIS. Alienists have recently taken into account far more than formerly the importance of the circulatory conditions in the brain in the development of psychoses. Barret[2] has published a study of the subject, and quotes largely from Alzheimer and Binzwanger, both of whom have contributed notably to it. Among the distinctive types of mental disease in which arteriosclerosis is recognized as an important pathological lesion are mentioned general paralysis, arteriosclerotic dementia, and, less certainly, senile dementia. Among the special symptoms in the different groups are mental fatigue, headaches, dizzy attacks, memory weakness, psychical phenomena with progressive dementia, fainting, apoplectiform seizures, and epileptiform convulsions. Aphasia, periods of excitement, and mental confusion are characteristic of some. In Barret's group of cases the loss of retentive memory was among the earliest symptoms. Later, inclination to fabulate, loss of judgment, disorientation, narrowing of the external interests, episodes of confusion and hallucinatory delirium, etc., followed more or less quickly. These briefly cited references indicate the kind and variety of symptoms as they present themselves to the alienist. The general clinician will find among them the extremes of many of the isolated symptoms presented by senile or even presenile arteriosclerotics who happily never advance to these unfortunate grades of cerebral disease.

ARTERIOSCLEROSIS IN THE YOUNG.

BY F. FREMONT-SMITH, A.B., M.D.,
ASSISTANT PROFESSOR OF PEDIATRICS IN THE GEORGE WASHINGTON UNIVERSITY; ASSISTANT PEDIATRIST TO THE UNIVERSITY HOSPITAL; ATTENDING PHYSICIAN TO THE HOSPITAL FOR FOUNDLINGS, WASHINGTON, D.C.

IN 1890 I attended, at St. Augustine, a male mulatto, aged thirty-two years, who was suffering from obliterative endarteritis with gangrene of the toes, requiring amputation. His skeletal arteries were sclerosed and beaded. He gave a history of luetic infection in his eighteenth year. As to other infections I am not able to speak.

During the period of service on this man my attention was drawn

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[2] Amer. Journal of Insanity, 1905, No. 1.